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A Review on Psoriasis - Types and Treatment

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ABSTRACT:

Psoriasis is an autoimmune disorder which is categorized as non-communicable disease negatively impacts the quality of life. The prevalence of this disease ranges between 0.09% and 11.4% among countries. Psoriasis typically affects the skin, causing skin cells to regenerate in rapid than usual. It develops into scaly patches, sores and causing inflammation in epidermis. There are several types of psoriasis which includes plaque, inverse, guttate, pustular, erythrodermic, psoriatic arthritis and nail psoriasis. Among these plaque psoriasis is the most commonest among individuals. Various comorbidities like hypertension, cardio vascular disorders, type 2 diabetes have been identified with psoriasis. Pathogenesis involves the hyperproliferation of keratinocytes which is induced by T cells, macrophages and dendritic cells. Diagnosis of affected skin includes epidermal thickness, scaling, redness which is scored for their disease progression level. Phototheraphy, coal tar, treating with corticosteroids have been found to be the early medications for psoriasis management. But more side effects of these medications have been reported in various cases, so to improve the patient's quality of life various plant extract based treatment have been proposed to be the future prospective for psoriasis treatment. This article briefly describes the variant types of psoriasis with its clinical manifestation, diagnostic criteria, immunology and various plant extracts which have been used in treating this disease.

Keywords: Psoriasis, keratinocytes, T cells, epidermal thickness, hyperproliferation.

INTRODUCTION:

An inflammatory skin condition that is immune-mediated and chronic, psoriasis is linked to a number of morbidities, including psoriatic arthropathy, psychiatric, cardiovascular, and hepatic disorders(1). The World Health Organization recognized psoriasis as a major non-communicable disease in 2014 and emphasized the distress caused by incorrect diagnoses, insufficient treatment, and stigmatization of the condition. According to the Global Burden of Disease Study, psoriasis caused at least three times as many disability-adjusted life years (DALYs) as inflammatory bowel disease in 2016 (5.6 million DALYs across all age groups)(2). Considered an autoimmune condition, psoriasis is influenced by both environmental and hereditary factors. The disease's name comes from the Greek word "psora," which meaning "itch." Psoriasis is a dry, inflammatory, unsightly skin condition that is non-contagious and can affect a person's entire body. It is primarily hereditary and is distinguished by erythematous, scaly, strongly marginated plaques that appear in a comparatively symmetrical pattern. The scalp, finger and toe tips, palms, soles, umbilicus, gluteus, under the breasts and genitalia, elbows, knees, shins, and sacrum are the areas most frequently impacted. This illness has a propensity to recur and is chronic in nature(3).

Erythema, scaling, epidermal hyperplasia, and leukocyte infiltration of the dermis and epidermis are the hallmarks of psoriasis, a chronic inflammatory skin condition. The pathophysiology of psoriasis is thought to be caused by the premature maturation of the skin's keratinocytes, which is brought on by a series of inflammatory mediators in the dermis that originate from dendritic cells, macrophages, and T cells and significantly contribute to the disease's exacerbation(4). Psoriasis is a primary keratinocyte condition, according to what we learned as dermatology residents. We discovered that the keratinocytes had gotten out of the regular cell cycle regulation and that they were often describing the illness to patients and to one another using a cancer model. In order to maximize keratinocyte DNA synthesis exposure to the medication, we employed a split dosing approach (every 12 hours for a total of 3 doses

per week) based on thorough DNA synthesis studies using antimetabolites like methotrexate to disrupt the cell cycle. Cyclic adenosine monophosphate was believed to be essential at the early stages for controlling keratinocytes and causing psoriasis(5). Researchers worldwide are looking for novel, safer, and more effective medications made from natural resources because the synthetic medications that are recommended to treat psoriasis have a number of negative side effects(6). In poor nations, traditional medicine plays a significant role in the healthcare system. The affordability and accessibility of traditional medicine are the reasons for its reliance. India formally acknowledges the therapeutic use of more than 3000 plants. Over 6000 plants are said to be used in traditional, folk, and herbal medicine in India, which accounts for over 75% of the Third World's medical requirements(7).

TYPES OF PSORIASIS:

Based on a number of characteristics of psoriasis, including the age of beginning, degree of skin involvement, morphologic pattern, and primary involvement of a particular anatomic site of the body, the disease spectrum or clinical phenotypes have been defined(8).

There are seven primary types of psoriasis:

- Plaque-type psoriasis or **Psoriasis vulgaris** (common type)
- Guttate psoriasis (small, drop like spots)
- Inverse psoriasis (in the folds like of the underarms, navel, and buttocks)
- Pustular psoriasis (pus-filled, yellowish, small blisters)
- Erythrodermic psoriasis (red, itchy, scaly skin plaques)
- Nail psoriasis (damaged, split, or lifted nails)
- Psoriatic arthritis (arthritis linked with psoriasis).

PLAQUE PSORIASIS:

Plaque is the most prevalent kind of psoriasis, characterized by dryness, itching, and elevated skin areas covered in scales. The most common locations are the lower back, elbow, knees, and scalp. The color of patches varies according on the skin's characteristics. As the damaged area of the skin layer heals, post inflammatory hyperpigmentation may cause transient changes in a person's color look, especially on dark or black skin(9,10).

The elbows, knees, lumbosacral region, intergluteal cleft, and scalp are the areas that are most commonly affected by this kind, which is distinguished by well-defined plaques with a loosely adhering silvery-white scale(11).

In psoriasis patients, the Koebner phenomenon—new lesions that develop after direct cutaneous trauma—occurs. Another well-known aspect of plaque psoriasis is the Auspitz phenomenon, in which a little disturbance of the lesion's outer layer causes pinpoint bleeding(8). Small pinpoint papules are often the initial stage of early lesions, which rapidly exhibit scaling. The first papules develop into bigger components and come together. An inverse type emerges in intertriginous locations when scaling is modest, avoiding the previously indicated typical sites(11).

GUTTATE PSORIASIS:

Children and young adults are commonly affected by this kind of psoriasis. Usually occurring following streptococcal infections, lesions show as tiny droplets at first, and less commonly as squamous psoriatic papules. The HLA Cw6 gene is most commonly linked to this kind of psoriasis. Anti streptolysin titers are frequently high. Lesions usually go away on their own as the illness progresses. Typically, lesions appear on the face, head, proximal portion of the limbs, and trunk. Usually, they regress in three to four months. Lesions can occasionally get larger and resemble psoriatic plaque(12).

Small erythematous plaques appear suddenly in guttate psoriasis, a variation of the condition. It often affects kids or teenagers and is frequently brought on by tonsil infections with group-A streptococci. Approximately one-third of those with guttate psoriasis will go on to develop plaque psoriasis(10).

INVERSE PSORIASIS:

Psoriasis that is confined in skinfolds is termed flexural or inverted psoriasis. Moisture and friction in skin creases do not cause squamous lesions. Lesions manifest as brilliant red, symmetric, infiltrative, fissured plaques with distinct features(12).

This type of psoriasis is characterized by smooth patchwork of inflamed skin that gets worse with perspiration and friction. It typically affects the folds of the skin around the breasts, buttocks, and crotch. Typically, fungal infections cause this kind of psoriasis(13).

PUSTULAR PSORIASIS:

The generalized form of pustular psoriasis manifests as a recurring, systemic sickness; the palmoplantar type manifests as a locally centered disease that mostly affects the palm and sole; or it manifests as acrodermatitis in the nail beds or fingers. Even though these disorders are uncommon, the severity and repercussions should not be disregarded. With the potential to have life-threatening consequences, such as a medical emergency of the generalized pustular form of psoriasis when it manifests as a flare-up or acute episode(13).

Hallopeau's acrodermatitis continuous with pustulosa palmoplantaris (PPP). PPP only affects the palms and soles of the hands and feet, but ACS is more widely distributed at the points. Multiple, sterile pustules that coalesce are a hallmark of pustular psoriasis.

There are two types of pustular psoriasis: localized and widespread. Psoriasis pustulosa palmoplantaris (PPP) and acrodermatitis continua of Hallopeau are two different localized manifestations that have been identified. Both conditions impact the hands and feet; ACS is more distally positioned at the tips of the fingers and toes and affects the nail apparatus, whereas PPP is limited to the palms and soles(10).

The generalized pustular form of psoriasis, a systemic inflammatory chronic illness, typically coexists with fever and malaise. In patients with widespread pustular psoriasis, there are many sterile pustules on the body's surface, accompanied with scattered erythema and swollen extremities. Given that generalized pustular often recurs throughout life, there may be potentially fatal circumstances. With the aid of current findings regarding the underlying genetic molecular foundation of various situations, clinicians and researchers are receiving significant advancements in the approach towards the pathomechanism of generalized pustular knowledge(13).

ERYTHRODERMIC PSORIASIS:

An estimated 1% to 2.25 percent of psoriatic individuals have erythrodermic psoriasis (EP), a severe and uncommon form of psoriasis vulgaris(14). Over 90% of the body's surface is erythematous and inflamed in erythrodermic psoriasis, an acute disorder. Any kind of psoriasis can develop erythroderma, which needs immediate medical attention(10). Generalized hypertension, dyslipidemia, obesity, and decreased glucose tolerance are the main causes of the erythrodermic phase(11).

EP may be linked to systemic symptoms as fever, tachycardia, lymphadenopathy, arthralgia, and exhaustion. More often affecting the fingernails than the toenails, nail abnormalities are very common in EP and can range from mild pitting to severe onychodystrophy. Fever, tachycardia, exhaustion, malaise, chills, dehydration, lymphadenopathy, arthralgia, myalgia, insomnia, sweats, diarrhea, constipation, weight fluctuations, allodynia, and infrequently high output heart failure (caused by excessive water loss and edema) and cachexia are other systemic symptoms that patients may experience(14).

NAIL PSORIASIS:

Psoriatic inflammation can also impact the specialized dermal appendages known as nails. More than half of psoriasis sufferers are said to have nail psoriasis, and 5–10% of people may have it as their sole psoriasis symptom(10). These are linked to scalp involvement and arthritis. Clinical signs and symptoms include subungual hyperkeratosis, onycholysis, severe onychodystrophy, pitting, yellowish discolouration, and paronychia(11). There are currently few and sometimes ineffective therapy options for nail psoriasis. While nail cholysis, or the separation of the proximal portion of the nail from the nail bed, is the most prevalent anomaly in psoriasis, nail pitting is the most distinctive trait.

The formation of "oilspots" or orange-yellow varnish beneath the nail plate indicates the involvement of the nail bed. Additionally, the nail plate may grow thicker, dystrophic, discolored, and yellow, and keratinous material may accumulate beneath it; this condition is referred to as subungual hyperkeratosis(8). Joint involvement is linked to psoriatic nail involvement, and nail symptoms are present in up to 80% of PsA patients(10).

PSORIATIC ARTHRITIS:

Psoriasis combined with inflammatory arthritis (spondylitis and/or peripheral arthritis) and often a negative serologic test for rheumatoid factor is known as psoriatic arthritis(15). Psoriatic arthritis is an inflammatory, long-term autoimmune-mediated condition that mostly affects the axial skeleton's joints and entheses. It is linked to a higher risk of dying from cardiovascular disease(13). Nearly equal distribution of males and females, asymmetrical peripheral arthritis affecting a few small joints, involvement of DIP joints, sausage digits, arthritis mutilans, ankylosing spondylitis, gout-like onset, and a higher incidence of nail involvement than in simple psoriasis are some of the disease's clinical features. The rash may appear alongside arthritis or, conversely, it may occur before or after joint dysfunction(15). There are additional characteristics that assist differentiate PsA from other types of arthritis in addition to the patterns of arthritis. PsA is distinct from Heberden's nodes of osteoarthritis due to the presence of inflammatory distal joint pathology. In individuals with RA, distal interphalangeal joint involvement is uncommon. The diagnosis becomes relatively clearer when distal joint involvement occurs in fingers that have shown nail alterations. Dactylitis, or inflammation of the whole digit, is common in PsA patients, however it can also happen to those who have Reiter's disease. It is believed that inflammation of the individual joints and the tendon sheaths of the afflicted digits causes dactylitis(16).

IMMUNOLOGY :

The immunology of psoriasis is manifested by IL-17 & IL-23 pathway. Type 17 T-cells, or T17 cells, are IL-17-producing T-cells that generate IL-17A and IL-17F3 in psoriasis skin after being activated by IL-23 from inflammatory dendritic cells(17). Keratinocytes increase cellular immunity to sustain prolonged T-cell activation by producing a variety of "feed-forward" inflammatory mediators in response to IL-17, including IL-36γ. Nearly all of the phase 3 clinical trials assessing the effectiveness of IL-23 and IL-17 blockades have measured the improvement of psoriasis 3 to 4 months after the first injection, and the high efficacy of biologic agents targeting IL-23 or IL-17 for psoriasis treatment has strongly supported the roles of IL-23 and IL-17 as key drivers of psoriasis inflammation(1). Type 1 (Th1) and type 17 (Th17) T helper cells are activated to create a cellular form of immune response by the IL-12 and IL-23 secreted by the antigen-presenting cells in the skin(18). The development of a state of chronic inflammation, altered hyperproliferation of the epidermis, apoptosis, differentiated mechanism, and neo-angiogenesis—all brought on by various cytokines, including tumor necrosis factor (TNF) α—are the causes of the cutaneous findings seen in this disease(9).

PATHOGENESIS:

The intact epidermal layer, which is made up of the stratum basal, stratum spinosum, stratum granulosum, stratum lucidum, and stratum corneum, serves the primary purpose of protecting the skin. The epidermis is composed of four cell types: Merkel cells, Langerhans' cells, melanocytes (8%), and keratinocytes (90%). In the stratum basal, new keratinocytes develop during the epidermal cell cycle. They then move toward the surface, where they accumulate keratin before dying when they reach the stratum corneum. Psoriatic lesions also have thicker epidermises (three to five times normal), dilated blood vessels, and infiltrating inflammatory cells, including neutrophils. The normal cell cycle takes approximately four weeks, but in psoriasis, this process is accelerated; keratinocytes in the basal layer divide every 1.5 days, and they reach the surface in just about four days. This results in non-keratinized stratum corneum and abnormal cell buildup, which causes scaly skin(2). While the exact cell responsible for the fundamental malfunction in psoriasis is still unknown, a number of potential possibilities have been put up, such as endothelial cells, fibroblasts, and keratinocytes. Abnormal keratinocyte differentiation, hyperproliferation, and the infiltration of inflammatory components into the skin are the main pathogenic anomalies in psoriasis patients. Furthermore, new research suggests that keratinocytes isolated from psoriatic plaques have a pattern that postpones apoptosis onset and prolongs longevity(19).

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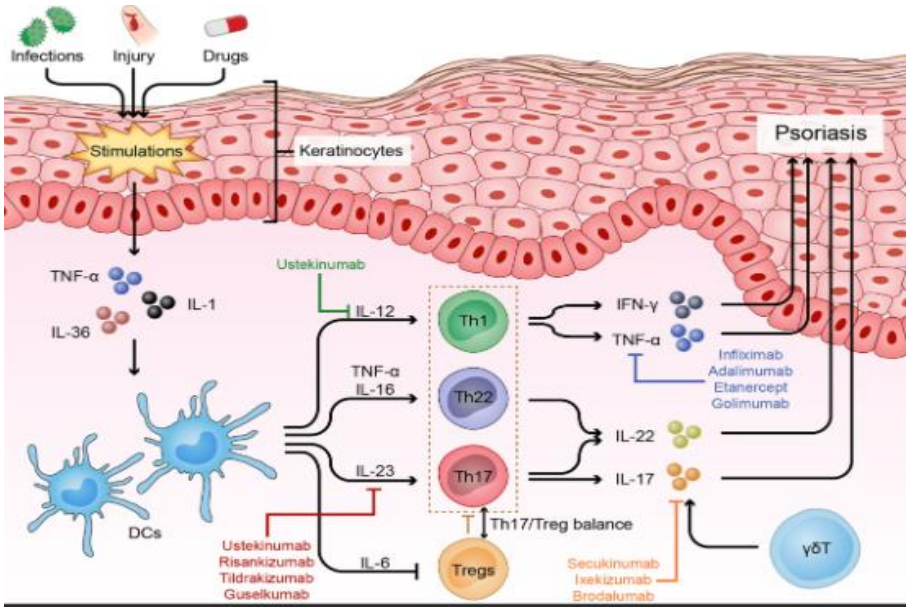

Figure 1: Signalling pathways of Psoriasis

Psoriasis was once thought to be a skin condition defined by hyperproliferation and aberrant keratinocyte differentiation. However, recent studies have confirmed that psoriasis is actually an autoimmune skin disease with T-cell-mediated responses. An imbalance between CD4+ T-cell subsets is one factor that causes both hyperkeratosis and parakeratosis. Numerous T-cell subsets, including regulatory T cells (Treg), T-helper (Th)1, Th2, and Th17, are implicated in the immunopathogenesis and chronic inflammatory response of psoriasis. Patients with psoriasis have been shown to have an increase in Th1 and Th17 cells and a reduction in Th2 and Treg cells. Therefore, preventing pathogenic T-cell activation helps to ameliorate clinical and histological conditions, such as excessive T-cell infiltration and aberrant K16 expression(20).Bone marrow transplantation may also cause psoriasis(21). The most recent discovery and focus has been on a novel inflammatory pathway that involves Interleukins 12 and 23(5).

FUNCTION OF TH17 CELLS IN INFLAMMATORY DISEASES AND PSORIASIS:

Th17 cells are a kind of CD4+ T lymphocyte that produces IL-17, a cytokine associated with a variety of chronic inflammatory disorders such as psoriasis and palmoplantar pustulosis. These cells are unique from Th1 and Th2 cells and play an important role in immune response and inflammation. T cells that produce IL-17 take role in immune surveillance in healthy skin. Inflammation is caused by an imbalance of Th17 and regulatory T cells. In psoriasis, streptococcal extracts can activate circulating effector memory T cells, causing them to generate Th17 cytokines that increase keratinocyte growth. This connection generates a positive feedback cycle in which activated keratinocytes stimulate T cell IL-17 production. In particular, IL-17A is the primary isoform involved in the pathogenesis of psoriasis. Th17 cells in psoriatic skin are additionally made up of CD8+ T cells and innate immune cells(21).

ROLE OF IL 23 IN PATHOGENESIS:

IL – 23 which is composed of IL -23p19 and IL – 12p40 is found to be the major cytokine in the pathway of psoriasis. It is majorly produced by dendritic cells, macrophages, monocytes and binds to its receptors like natural killer T cells, memory T cells. In psoriatic condition elevated amount of IL – 23p19 and IL – 12p40 is observed which contributes to the inflammation by promoting acanthosis and hyperkeratosis through the TNF- α (pro – inflammatory cytokine) production, which is a major cause of psoriasis(21). The pathogenesis takes place in two stages: start and maintenance. External stressors, such as trauma, infections, and stress, cause keratinocytes to produce self-DNA and LL-37 (cathelecidin – anti microbial peptide) stimulating immunological responses. This causes keratinocyte proliferation, erythema, and scaling by inducing plasmacytoid dendritic cells to create cytokines that activate T-cells. These T-cells then return to the skin and release inflammatory cytokines including IL-17 and TNF-α. Pro-inflammatory cytokines are also produced by keratinocytes, and Th17-cells, which were discovered around ten years ago, are a major source of IL-17A. This inflammatory cycle, which includes neutrophil and macrophage recruitment, adds to the severity and symptoms of persistent psoriasis(22).

MECHANISMS OF ENVIRONMENTAL RISK FACTORS' IMPACT ON PSORIASIS

UV Light and the Management of Psoriasis

UV-B radiation has been used for decades to treat psoriasis due to its ability to promote vitamin D production, which regulates keratinocytes and immune cells. UV-B exposure, particularly at 311 nm, induces apoptosis in keratinocytes, reduces Th17 cell growth, and decreases inflammation, leading to effective lesion clearing. This makes UV-B therapy a cost-effective and successful treatment for mild psoriasis. Factors like UV exposure, sunscreen use, and latitude can influence its effectiveness (20).

EXACERBATION OF PSORIASIS AND MEDICATION:

Certain medications, including antivirals, antidepressants, anti-proliferative like Imiquimod, anti-hypertensives, and TNF-α inhibitors, can trigger psoriatic lesions. TNF-α and INF-α play key roles in psoriasis inflammation, with TNF-α medication increasing INF-α expression. Imiquimod is particularly significant in inducing psoriasis through the INF-α signaling pathway. While anti-TNF-α biologics can cause psoriasis in a small group of patients, this highlights the importance of risk assessment and safety management when prescribing these drugs (20).

PSORIASIS RISK WITH SMOKING:

Smoking and alcohol use are both associated with worsening psoriasis. Smoking can decrease the effectiveness of TNF-α inhibitors, speed up the development of psoriatic arthritis, and lead to worse outcomes due to its impact on immune cell and keratinocyte interactions. Additionally, smoking increases the risk of cardiovascular diseases and other comorbidities in psoriasis patients. Alcohol, while its role in initiating psoriasis is debated, is known to exacerbate the condition by increasing inflammation and promoting keratinocyte formation. Excessive alcohol use, especially beer, can worsen psoriasis severity, though the exact mechanisms need further research(20).

DIET, WEIGHT LOSS AND MANAGING PSORIASIS:

According to research, weight loss and healthy eating habits can improve psoriasis symptoms. Obesity, which is associated with a pro-inflammatory state, increases the likelihood of psoriasis incidence and severity, with research indicating that a higher BMI corresponds with worsened psoriatic outcomes. Patients may benefit from diets high in omega-3 fatty acids, including fish oil, vitamin B12, vitamin D, and selenium. Some research suggests that a gluten-free diet may have a therapeutic impact. While weight reduction and dietary modifications show promise in treating psoriasis, further study is needed to prove their clinical efficacy in disease management(20).

CLINICAL MANIFESTATION:

The clinical manifestations of psoriasis are quite common. Primary skin lesions might include papules, plaques to pustules, and macules. Skin and nails may not be the only areas affected by the condition. The symptoms varies accordingly to various types and site of location. Plaque psoriasis, is distinguished by distinct salmon pink plaques with silvery-white scales that are frequently distributed symmetrically. It mostly affects the scalp, trunk, and extensor surfaces (knees, elbows)(23).Guttate psoriasis is characterized by an abrupt symmetrical eruption of drop-like papules/plaques mostly affecting the trunk and limbs, sometimes preceded by streptococcal infection (but not always)(2). Pink papules that eventually turn scaly are the early sign of this kind of psoriasis Guttate psoriasis patients might later upon develop plaque psoriasis(23,24). Erythrodermic psoriasis (EP) is characterized by widespread erythema, edema, pruritus, ill-defined psoriatic plaques, scaling, hair loss, and occasionally exudative lesions or desquamation. Onychodystrophy and pitting of the nails are frequent(14).Pustular eruptions on the fingers and toes, which can extend proximally to the dorsal parts of the hands and feet are the signs for pustular psoriasis(8). Nail psoriasis is found on fingernails and toenails, it begins as several pits and occasionally develops into thicker, crumblier, yellowing nails; it may peel off(11,23).An inflammatory arthritis may develop in around 30% of patients, and psoriatic disease patients may also occasionally develop uveitis and inflammatory bowel disease. Additionally, the appearance might differ significantly from patient to patient. The skin manifestations might be monomorphic, polymorphic, restricted, or vast, and they can present at any age. Because of this variety in clinical presentation, psoriasis may be considered as a spectrum of disease, and there are currently no clear diagnostic criteria for the condition(8).

DIAGNOSIS :

The diagnosis of psoriasis is often simple and is based on clinical symptoms (skinrash, nail changes, joint involvement). Sometimes patients have non-specific skin indicators such mild scalp scaling, isolated flexural erythema, or orgenital lesions, or they appear with atypical skin lesions that need to be separated from tinea, mycosis fungi, discoid lupus, or seborrhoiec dermatitis. A skin biopsy may occasionally be necessary, and careful examination of fall sites may show traits that have been disclosed and are diagnostically valuable. Although guttate psoriasis, flexuralor "inverse" forms (bodyfolds), sebopsoriasis, erythrodermic psoriasis (total body redness and scaling), and pustular psoriasis (localized or generalized palmar disease) are morphological variations of chronic plaque psoriasis. Sometimes combinations of the various categories arise in the same patient either simultaneously or gradually over time(25).

While topical treatments are often used to treat mild cutaneous psoriasis, systemic or phototherapy may be necessary for moderate-to-severe cases. More than 5 to 10 percent of the body surface area (BSA) is often affected by moderate-to-severe illness. Regardless of BSA, psoriasis can be severe when it affects a person's physical, social, or psychological well-being or when it just affects certain regions, such as the face, genitalia, palms, or soles(26).

MANAGEMENT AND TREATMENT OF PSORIASIS:

There are currently only suppressive treatments available for psoriasis; there is no cure. Patients with mild illness frequently manage their condition by avoiding triggers, however therapy is sought when symptoms including discomfort, itching, or cosmetic issues interfere with everyday living. The objective is to lessen the severity of psoriasis so that it has less of an impact on job, social life, and overall health. Even for individuals with up to 10% skin involvement, topical therapies remain the first-line treatment for stable plaque psoriasis. Systemic treatment, however, can be necessary for more severe instances (greater than 20% body involvement)(27). Emollients, keratolytic drugs, anthralin, tars, topical corticosteroids, vitamin D3 analogues, retinoids, methotrexate, cyclosporine, tacrolimus, and psoralens plus phototherapy are among the treatments available for psoriasis. Topical therapies are often the initial line of treatment for mild to moderate psoriasis, while a more aggressive approach may be required for severe instances. The goal of these therapies is to control symptoms and minimize the severity of the illness(28).

MOISTURIZERS, EMOLLIENTS, AND KERATOLYTIC AGENTS IN THE TREATMENT OF PSORIASIS:

By creating an occlusive layer that stops water loss and enables the skin to recharge, emollients and moisturizers aid in hydrating and soothing dry, flaky skin. In addition to enhancing skin pliability and minimizing scaling, this may also have antipyretic and vasoconstrictive benefits(29). They should be freely administered up to three times a day and are frequently used as a pretreatment for psoriatic plaques. Folliculitis and contact dermatitis are side effects. Salicylic acid and other keratolytic medicines help to reduce hyperkeratosis and remove scales. Lower quantities of salicylic acid are more effective because they reduce the skin's corneocyte collaboration(28)(30).

COAL TAR TO TREAT PSORIASIS:

For ages, people have used coal tar to cure psoriasis. It acts by lowering epidermal cell mitosis, irritation, and sebum production. It works extremely well for relieving severe itching and curing guttate psoriasis. Coal tar comes in a variety of forms, including lotions, gels, and conditioners; the most effective concentration is 5%. However, it has the potential to induce skin irritation, acneiform outbreaks, folliculitis, and contact dermatitis, particularly in people who have been exposed to tar. It also has a phototoxic impact that exacerbates sunburns. Because of the risk for irritation, coal tar is not advised for erythrodermic or pustular psoriasis(27,28).

ANTHRALIN TO TREAT PSORIASIS

One topical medication for psoriasis that has demonstrated effectiveness in removing plaque is anthralin. A short-contact regimen, in which anthralin is applied for one hour and then withdrawn, has become the favored procedure because to its efficacy and fewer adverse effects when compared to standard overnight treatments. This approach permits anthralin to enter the lesional skin without damaging the perilesional regions. Anthralin reduces inflammation and slows down the development of new skin cells. With fewer side effects, it works just as well as topical corticosteroids. Anthralin can help with mild to moderate psoriasis, however it can also irritate and discolor skin. Because it can discolor skin and clothing, patient acceptability may be limited despite its efficacy(27,28).

TOPICAL CORTICOSTEROIDS:

Topical corticosteroids can help treat psoriasis by lowering inflammation, itching, and scaling. They bind to glucocorticoid receptors, exerting anti-inflammatory, antiproliferative, and immunosuppressive properties. They work better and have fewer

negative effects when taken with vitamin D analogs or keratolytic medicines such as halobetasol propionate and tazarotene. These combination therapies can also be given twice a week proactively as lesions improve, providing better outcomes while reducing side effects(31,32).

PHOTOTHERAPHY:

PUVA, broadband UV-B, and narrowband UV-B are examples of phototherapy used to treat moderate-to-severe psoriasis. Because of its greater effectiveness, safety, and decreased risk of skin cancer, narrowband UV-B is recommended(29). Although PUVA, which uses UV-A and psoralens, is effective, it has a long-term cancer risk. Photoaging, blistering, erythema, and pruritus are some of the side effects of phototherapy. Despite its convenience, home phototherapy may have space and insurance restrictions(31,32).

TNF- α INHIBITORS:

TNF-inhibitors, such as etanercept, infliximab, and adalimumab, suppress the inflammation-causing cytokine TNF-alpha. After 8–16 weeks of therapy, a response is observed. They treat moderate-to-severe psoriasis but can induce serious adverse effects such as infections, hepatitis reactivation, TB, lupus, and demyelinating diseases(31,33).

IL-23 INHIBITORS:

Tildrakizumab, Risankizumab, Guselkumab, and Ustekinumab all effectively suppress IL-23 in psoriasis. The PASI response rates for Risankizumab and Guselkumab are high. Mild side effects include tiredness and infections(31). The Inhibitors of IL-17 Targeting IL-17, Secukinumab, Ixekizumab, Bimekizumab, and Brodalumab provide quick relief for psoriatic arthritis and plaque psoriasis. Side effects include infection, mucocutaneous candidiasis, and potential mood swings, however efficacy is high(17,31).

GREEN ALTERNATIVES FOR PSORIASIS TREATMENT:

BOTANICAL NAME	FAMILY	PART USED	EXTRACTION	PHYTOCONSTITUENT	INDUCING AGENT	OBSERVATION	REFERENCE
Cassia tora L.	Fabaceae	Leaf	Ethanolic extract - 400 mg / Kg	Flavanoids	UV – B	Munro's microabscess, elongation of rete ridges, and capillary loop dilation	(34)
Solanum xanthocarpum	Solanaceae	Stem	Ethanolic extract - (Gel at 2.5, 5 and 10%) as well as orally (at 100, 200 and 400 mg/kg p.o.)	Phenols, tannins, flavonoids, alkaloids and carbohydrates.	Imiquimod	TNF-α, IL-1β, IL-6 and IL-17	(35)
Psorospermum febrifugum Spach	Hypericaceae	Leaf and Stem	Aqueous and ethanolic extract - 200 and 400 mg/kg	-	2, 4-dinitrofluoro benzene (DNFB)	Haematological and Haemagglutinin antibody	(36)
Woodfordia fruticosa (L.) Kurz	Lythraceae	Flower	Ethanolic extract - 0.05% and 0.1% (w/w) ointments	Flavonoids, glycoside, phenolics, and tannins	Complete Freund's adjuvant & formaldehyde mixture (1:10 ratio)	Phenotypic (redness, erythema, and scales) and histological features (epidermal thickness)	(37)
Cassia fistula L.	Fabaceae	Fruit	Methanol extract –	-	PPD-induced psoriasiform	Histometric analysis of	(38)

			cream at 2.5, 3.75, 5.0, 6.25, and 0% (w/w)			orthokeratosis and relative epidermal thickness.	
Gynura pseudochina (L.) DC.	Asteraceae	Leaf	Ethanolic extract	Phenolic acids, flavonoids, xanthone derivative, phenylpropanoid, phenolic glycoside compound and glycerol-phospholipid.	-	Non-TNF-α and TNF-α stimulation. IN VITRO – HacaT cells	(39)
Caesalpinia bonduc (L.) Roxb.	Caesalpiniaceae	Leaf	Butanol and water fraction – 250mg/Kg and 500mg/Kg	-	Mouse tail test	Epidermal thickness and percentage orthokeratotic values. Antiproliferant assay and lipoxygenase inhibition assay.	(40)
Memecylon malabaricum Cogn.	Melastomataceae	Leaf	Aqueous extract & hydroalcoholic extract	-	-	Orthokeratosis and thymidine phosphorylase inhibition assay	(41)
Pongamia pinnata	Fabaceae	Leaf	Hydroalcoholic leaves extract	-	Imiquimod	Collagen content, angiogenesis, keratinization, fibroblast proliferation	(42)
Melissa officinalis ssp.	Lamiaceae	Aerial parts	Non polar and polar extracts - using dichloromethane and methanol	-	Imiquimod	Histopathological assessment, measurement of TEWL and hydration	(43)
Thespesia populnea	Malvaceae	Bark	Ethanol and aqueous extract	Carbohydrates, glycosides, tannins, flavonoids, triterpenoids, phytosterols, proteins and lipids/fixed oils	UV Induced wound	Orthokeratosis	(44)
Mangifera indica	Anacardiaceae	Flower	Hexane, ethanol and olive oil.		Imiquimod	PASI Scoring	(45)
Ipomoea batatas	Convolvulaceae	Leaf	Aqueous extract	Phenols and Flavanoids	Imiquimod	Antioxidants with potential anti-psoriatic	(46)
Dillenia indica Linn.	Dilleniaceae	Fruit	Aqueous ethanolic extract (AEE), ethyl acetate	-	UV induced wound	Lipid peroxidation and Orthokeratosis	(47)

			extract (EAE) and petroleum ether extract.				
Psorospermum febrifugum Spach	Hypericaceae	leaf and stem bark extract	Aqueous and ethanolic extracts – 200mg/Kg and 400mg/Kg	-	2, 4-dinitrofluoro benzene (DNFB)	Anti-inflammatory, haematological and Haemagglutinin Antibody	(36)
Withania somnifera	Solanaceae	Seed	Supercriticalcarbondioxide(SC-CO2)extraction	-	TPA (12-O tetradecanoyl phorbol 13-acetate)	NFB, IL-6, TNF, MPO Assay	(48)
Woodfordia fruticosa (L.) Kurz	Lythraceae	Flower	Ethanolic extract - 0.05% and 0.1% (w/w) ointment	Flavonoids and polyphenols	Complete Freund's adjuvant (CFA) and formaldehyde mixture (1:10 ratio)	TPC, TFC, PSI and histological examination.	(37)
Psorospermum febrifugum	Hypericaceae	Flower	Alcoholic and aqueous Extracts – 200 mg/Kg and 400mg/Kg	-	2, 4-dinitrofluoro benzene (DNFB)	Ear thickness, Body weight	(49)
Citrus sinensis	Rutaceae	Peel	Aqueous extract and ethanol extract – cream (100mg/kg)	-	Perry scientific tail model	Epidermal thickness and percentage orthokeratosis.	(50)
Actinidia arguta	Actinidiaceae	Fruit	Distilled water	-	Imiquimod	Skin thickness and Interleukin (IL)-17A, Neutrophil Chemotaxis Assay.	(51)
Rubia cordifolia L.	Rubiaceae	Root	Ethanolic extract fractioned with hexane, ethyl acetate (EA), n-butanol and water		Tail model	Keratinocyte Proliferation	(52)
Phoenix dactylifera	Arecaceae	Seed	Methanol extract - 2% and 5% cream	-	Imiquimod	Histopathological, skin neovascularization and spleen index	(53)
Moringa oleifera L.	Moringaceae	Seed	Ethanol and water (95:5) v/v.	-	12-O-tetradecanoyl phorbol-13-acetate (TPA)	IL-12/IL-23 p40, IL-17A, IL-22 and IL-23 p19, markers such as profilaggrin and loricrin	(54)
Artemisia capillaris	Asteraceae	Whole plant	Ethanolic extract – cream 2%	-	Imiquimod	Intracellular adhesion molecule-1	(55)

						(ICAM-1), PASI.	
Annona squamosa	Annonaceae	Seed	Petroleum ether extract	Polyunsaturated fatty acids viz. linoleic acid and oleic acid	Oxazolone and olive oil (4:1)	Cytokines IL6, IL17, TNF-α, INF-γ, GMCSF, and infiltration of CD4 T cells	(56)
Dictamnus dasycarpus Turcz.	Rutaceae	Root	Ethanol	-	Imiquimod	Interferon (IFN)-γ and Interleukin (IL)-17	(57)
Givotia rottleriformis	Euphorbiaceae	Bark	Ethanol	Flavanoids	(UV-B)-induced	ANOVA	(58)
Scrophularia deserti	Scrophulariaceae	Aerial part	Methanol	Phenol and Flavanoid	Imiquimod	DPPH, IL-22, TNF-α,IL-17A, Histopathology	(59)
Ficus carica	Moraceae	Fruit	Methyl alcohol	Polyphenol and Flavanoid	Imiquimod	PASI, STAT-3 modulation	(60)
Ammi visnaga L	Apiaceae	Seed	Distilled water	-	UV- induced	Acute toxicity, Body weight, Bioserum analysis, Histopathology of liver and kidney.	(61)
Melissa officinalis ssp. altissima	Lamiaceae	Aerial parts	Dichloromethane and methanol	Polyphenols	Imiquimod	DPPH, PASI, TEWL,Histopathology	(62)
Psoralia corylifolia	Fabaceae	Seed	Ethanolic extract	Carbohydrates, triterpenes, fats & oils, resins, phenols, flavonoids, steroids and monoterpenes.	Mouse tail model	Sulphorhodamine B (SRB) assay, orthokeratosis	(63)

CONCLUSION:

Psoriasis, a major psycho – social disorders which affects the patients social, physical & mental state of quality of the life of individuals. This review emphasizes on clinical manifestation of psoriasis with their challenging aspects on diagnosis. Clinical manifestation of psoriasis involves the hard, scaly patches, epidermal thickenening, redness and inflammation. By cellular concept, there will be increased hyperproliferation of keratinocytes than usual cell growth. Immunology of disorder is manifested mainly by T helper cells of which Th 17 helper cells has been affected the most. IL- 23 proliferation causes IL- 17 cells to develop, hyper proliferation of these cytokines causes the cells to multiply rapidly causing thick epidermal scaly patches. These cytokines further develops visible sores, inflammation and causing the apoptosis of cells. Based on manifestation and location various types of psoriasis have been identified among which plaque psoriasis is the common type. Psoriasis induction methods like UV – B induction, DNFB, Imiquimod, Mannan, Oxazolone have been mentioned in many articles which causes the T cell hyperproliferation. Traditional methods includes topical application of corticosteroids like Clobetasol, Halobetasol which binds to glucocorticoid receptor and providing anti-inflammatory and anti-proliferative effect. Phototheraphy is also found to be effective against psoriatic lesions.

Various medications include TNF- α inhibitors, IL- 17 inhibitors, IL- 23 inhibitors have been implemented in treating the manifestation of psoriatic conditions. As these medication causes a long term side effect, green theraphy is required for the treatment of psoriasis – Plant extract based approaches. A number of plant varieties have been identified for curing psoriasis – leaf, stem, bark, fruit, seed, oil, root & as a whole part is used for the extraction process. A list of various plant extracts & family with their inducing agent have been discussed in this review. By emphasizing the need on plant based approach could be a upcoming tool for management of psoriasis treatment.

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